found that they arose as delicate fibrils extending into the surrounding tissue from the old, coarse fibres of the adventitial coat. The author believes, therefore, that these new elastic fibres found in carcinomata of the stomach take their origin from the pre-existing elastic tissue of the walls of the bloodvessels.—W. T. L.

Contribution to the Cytological Study of Tuberculous Pleurisies.—BARJON and CADE (*Arch. Gén. de Méd.*, August, 1902, p. 160). The authors report in detail a study of the types of cells found in the exudates from forty-three cases of tuberculous pleurisy. Of this number twenty-five were proven to be of tuberculous origin from autopsies, sero-diagnosis, or inoculation, while the remaining observations were made from cases probably tuberculous in origin, but without definite proof of it. In practically all the cases the predominant cell was the small lymphocyte, sometimes reaching as high as 65 per cent. to 98 per cent. of the total number of cells. Early in the disease polymorphonuclear leucocytes may be present even in large numbers, but as the process develops these cells almost entirely disappear. Ravant has found a constant small percentage of polymorphonuclear leucocytes, this being true even for the early stages of the disease. The authors account for their somewhat different results by the fact that their observations were made upon centrifugalized fluid, whereas Ravant defibrinated the exudate, and thus lost sight of many polymorphonuclear leucocytes which were caught in the fibrin clots. Barjon and Cade lay little stress upon the numbers of endothelial cells present in the exudate. They believe a high percentage of small lymphocytes in the pleural exudate is a constant occurrence in tuberculous pleurisy.—W. T. L.

HYGIENE AND PUBLIC HEALTH.

UNDER THE CHARGE OF

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The Cause of Beriberi.—In a "Discussion on Beriberi" (*British Medical Journal*, September 20, 1902, p. 830) PATRICK MANSON calls attention, first, to the importance of coming to an agreement as to the meaning of the term, which, he asserts, has been applied to a variety of other diseases. He believes that even the most experienced practitioners include several distinct forms of neuritis under the one term, and he holds up the history of malaria as a warning, calling attention to the fact that, before the cause of malaria was determined, many types of non-malarial fever were regarded as malarial. Beriberi being a multiple peripheral neuritis, and this condition being the consequence of a number of causes (arsenic, alcohol, ptomaines, etc.), it is not strange that where beriberi exists some confusion must occur. For his belief that beriberi is not arsenical neuritis, he gives a number of reasons, among which is the fact that tobacco, to which arsenic is intentionally added to give it a desired flavor, is not used in institutions, such as jails and schools, in which the disease is prevalent; and, moreover, in the treatment of the disease, arsenic is not uncommonly exhibited and causes no aggravation of the symptoms, which would occur if arsenic were the cause. Furthermore, the skin affections common in arsenical neuritis are not observed in beriberi. He holds that the great majority of cases of so-called malarial neuritis are in reality beriberi, and that the true malarial neuritis is very rare and never epidemic. He believes that beriberi is due to a toxin produced by a germ operating in some culture medium located outside the human body, and that the toxin enters the body neither in food nor in water, but through the skin or lungs. In support of his contention that the cause cannot be a germ living and multiplying in the body, he cites the fact that the most important measure in the treatment of the disease is removal of the patient from the place in which he sickened, which, if accomplished early, and provided the dose has not been overwhelming, is almost invariably followed by recovery. In proof of this statement, the details of a most interesting outbreak are given. The fact that the disease can be introduced into and spread in virgin country is given as proof that its cause is produced by a living germ. It was introduced by the Japanese into the Fiji Islands, by the Annamites into New Caledonia, and by others elsewhere. Thus far all trustworthy investigations have failed to demonstrate the presence of the germ within the human body. That the toxin does not enter the system in food he concluded long ago, by the process of exclusion and on epidemiological grounds. He has no sympathy with the rice theory, and brings forward cogent arguments against it. Proofs that the toxin is not conveyed in drinking water are too numerous to need mention, for it often happens that of two institutions side by side, supplied with the same water, one is the seat of the disease and the other is free from it. He concludes that since the disease is not caused by a germ operating directly in the human body, it cannot be passed as an infection directly from person to person; that being the result of a toxin generated by a germ outside the human body and not conveyed in food or water, it must be conveyed either by the air or through the skin, by contact or by means of some insect which inserts it under the skin, or by a combination of some of these ways; and that, unfortunately, we have nothing to show what the toxin is, nor what the germ is that produces it, nor how it gains access to the body.

Captain E. R. Rost disagrees most decidedly with Manson, and believes strongly in the alimentary origin of the disease. He relates that during an outbreak in a jail he observed that pigeons which lived in large numbers under the roofs of the jail buildings were affected by an epidemic disease, characterized by paralysis of the wings, which he attributed to a micrococcus which he found in the rice. Later, in Rangoon, he found in rice-water liquor and in mouldy rice a spore-bearing organism which was extremely resistant to high temperatures, exposure to 220° F. for nine hours being required to kill the spores. He found the same organism in the blood and in the cerebro-spinal fluid of a large number of beriberi victims. Cultures injected into fowls caused death, and the organism was found in the blood and cord. An exactly similar disease was produced in fowls by feeding them on fermented rice obtained in the rice-liquor shops and on mouldy rice from the lower bags of damp godowns, by intraperitoneal injection of rice-water liquor, by subcutaneous or intraperitoneal injection of the venous blood of beriberi patients, and by re-injection from fowls suffering from the condition produced. The blood of beriberi patients used in the same way in pigeons produced symptoms in from one to three weeks, followed by death. Of 390 patients treated for beriberi in one institution in one year, all were adults and but few were women, and Rost believes the disease to have been due to a liquor made from damaged rice according to a process which he describes. As some outbreaks cannot be traced to the use of rice liquor or to diseased rice, he deems it probable that rice is not the only cereal in which the cause of the disease can grow, and he believes, moreover, that it is possible for the disease to be communicated through fowls suffering therefrom.

Dr. L. W. SAMBON supports the rice theory, and points out the fallacies in certain observations which have been brought forward to disprove it. He believes that rice may be related to beriberi as the vehicle and not the cause of the infection. Paddy keeps sound for years, but the hulled rice is soon damaged by vegetable parasites. He cites Eijkman's statistics applying to 280,000 prisoners in Java, where one convict in thirty-nine fed on white rice has the disease, while but one in 10,000 fed on red rice acquires it. He believes that the specific agent may remain latent for a long time in the system, and thus accounts for the numerous outbreaks at sea and among people who have gone where the disease did not previously exist, and for the limitation of the infection to those who have introduced it. Infection does not spread in hospitals to other patients, and it does not attack nurses or attendants. Yet the disease is essentially a disease of collective buildings (institutions), a fact which, at first sight, might suggest contagion. Overcrowding may not be looked upon as a leading cause, for change in this regard appears to bring about no improvement. The disease breaks out just as often in new as in old buildings, and in modern battleships as well as in old wooden merchantmen.

RONALD ROSS brings forward additional evidence in support of the claim that arsenic is connected with the disease. He had already published the results of analysis of the hair of twenty-two patients with the disease, arsenic having been found in traces in that of seven, principally the most recent cases. The cases which yielded negative results were, with two exceptions, of from one month to two years' duration. He records, now, the analysis of samples from eight new cases, the three most recent of which yielded positive results. The negative cases were all of more than thirty-two days' duration.

Distribution of Sewage Bacteria in Drinking Water. — Concerning the alleged ubiquity of the colon bacillus and the importance of streptococci as indicators of the presence of sewage matters in drinking water, a valuable and instructive paper is contributed by C.-E. A. WINSLOW and MISS M. P. HUNNEWELL (*Journal of Medical Research*, December, 1902),